with a decreased output postoperative of 14 per cent., the urine showing a trace of albumin and many hyaline casts.

Comparing this with the results obtained from ether anesthesia, in 3 of the cases which were very nervous, the average ante-operative phthalein was 55 per cent., the postoperative phthalein, 54 per cent., or a difference of 1 per cent. as compared with the difference of 4.5 per cent. in the ether series of nervous patients.

SUMMARY AND CONCLUSIONS. Upon the whole, the average case after thirty-six hours postoperative shows very little change in kidney function as demonstrated by the phenolsulphonephthalein test, although 25 per cent. showed urinary changes, which in ten days time were again negative.

The functional activity of the kidney is depressed as the length of anesthesia is increased, while in short anesthesias the kidney might even appear to be stimulated to a slight degree.

As age increases the threshold activity of the kidney is lessened.

Nervous patients, anemic, obese, and arteriosclerotic patients as a rule, show some effect of their physical or psychic state on kidney function.

Preëxisting conditions of albuminuria have a tendency to decreased phthalein excretion, and those cases which have a decreased phthalein excretion, in the majority of instances, show effects of kidney depression as evidenced by careful urinary analysis, although these effects are only temporary.

For long anesthesias and apparently for nervous patients, gas and oxygen as an anesthetic seems to have the least irritating effect on kidney function, as demonstrated especially by urine examination.

AN UNUSUAL DISORDER OF THE CARDIAC MECHANISM RELIEVED BY SURGICAL OPERATION.

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FROM the two points of view the following case is of interest, as an example, (1) of an unusual type of disordered mechanism of the heart beat, and (2) of the efficacy of removing cryptic or frank sources of reflex damage to the cardiac mechanism.

History. A. R., a married woman, aged fifty-six years, was admitted to the surgical wards of the University Hospital for repair of cystocele and rectocele. On physical examination, however, the pulse was found to be so distinctly irregular that the operation was

postponed. The following pertinent facts were obtained from the past history and examination:

The patient has never had any cardiac symptoms further than slight attacks of palpitation for several years, associated with indigestion and flatulence. For the past two years she has noticed some slight dyspnea on exertion, but never any edema or chronic cough. She did not know that her pulse was irregular. The pelvic symptoms—pain in right side of abdomen and a palpable mass in the vagina—have been present for two years. She has had four healthy children and one miscarriage (probably from overexertion). Aside from the coupled beats and pelvic disorders, physical examination revealed nothing of importance. The heart was of average size and the sounds normal. At times there was a pulse deficit of 50 per cent., due to the second beat of each couple failing to reach the wrist, but this beat was always sufficient to raise the aortic valves. There was no congestion, edema, or other signs of myocardial weakness. The urine showed a normal specific gravity, a faint trace of albumin, but no casts. Blood examinations were negative.

An electrocardiogram taken at this time revealed (Fig. 1) a condition of coupled beats, that on superficial examination might be mistaken for nodal rhythm coupled with regularly recurring extrasystoles arising from a part of Tawara's node other than that responsible for the nodal rhythm. The arrhythmia was later considered, however, to be due to auricular extrasystoles coupled with normal beats, both being followed by an unusually short conduction time. The basis for these interpretations will be discussed later. The atropin test (2 mgms. hypodermically) failed to affect the "coupled" rhythm, although the cardiac rate was raised from 92 to 118 beats per minute. Digitalis medication (0.67 c.c. of the tincture four times a day) succeeded after six days in lengthening the *P-R* interval to 0.16 second, but the premature contractions continued to appear almost constantly in the form of "pulse bigeminus."[1] (It is perhaps noteworthy that the *P-R* interval of the first cycle of each pair began to lengthen after three days of digitalis, whereas the second cycle required eight more doses before it was affected.)

As the arrhythmia had been found not to be of serious import, the contemplated surgical operation was performed with uneventful success. The pulse-rate did not go beyond 106 at any time during the operation, and was noted as regular during the period of anesthetization. This regularity continued during convalescence, and an electrocardiogram taken at that time showed that a normal mechanism existed (Fig. 2). As the influence of digitalis wore off the shortening *P-R* interval did not return to its former position beyond

[1] The influence of digitalis on the form of the T-wave (Cohn, Jour. Exp. Med., 1915, xxi, 593) is fairly well demonstrated in this case.

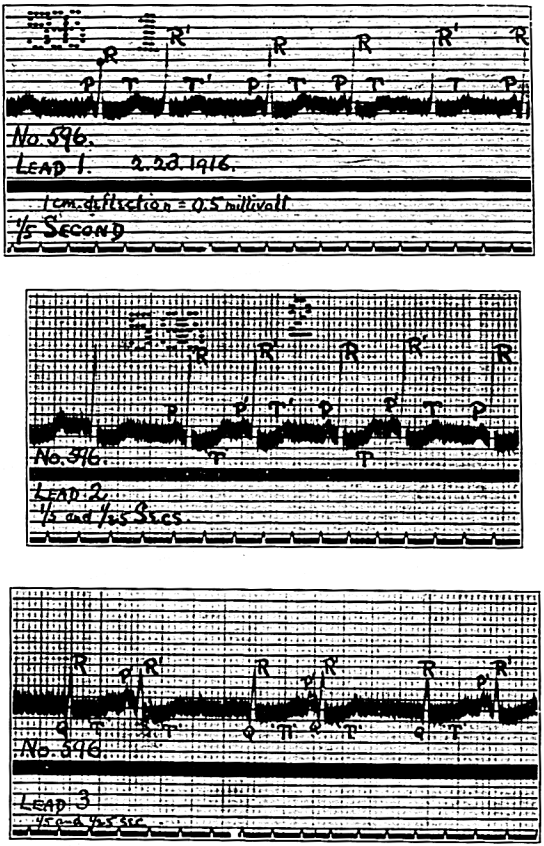

Fig. 1.—Electrocardiograms from the three customary leads, showing shortened *P-R* interval and "pulsus bigeminus," due to recurring auricular extrasystoles. Note that the *P* of the second cycle of each pair is of slightly different form from that of the first. In Lead 1 the ectopic P-wave and in Lead 3 the normal P-wave are not plainly distinguishable, but were present in other records taken during the period of bigeminy. The *P-R* interval is 0.10 and 0.09 second respectively. (Normal *P-R* interval 0.12 to 0.17 second.)

the shorter normal limits, and when the patient was last seen the ectopic beats had not reappeared. The patient continued in good

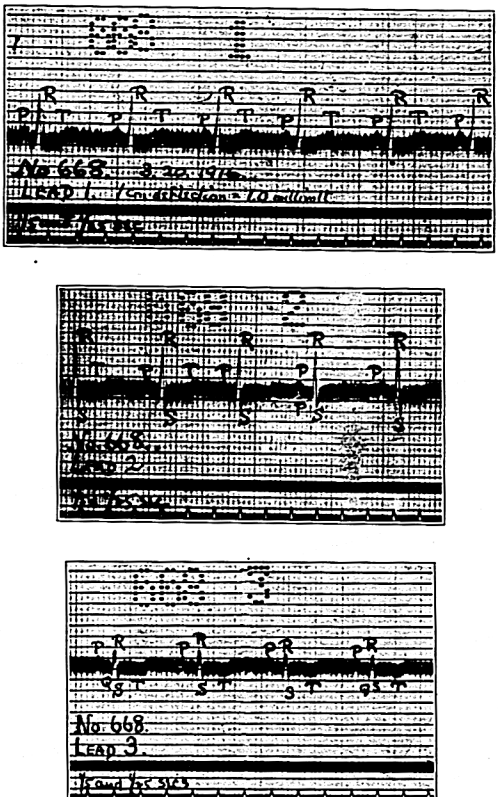

FIG. 2.—Electrocardiograms showing normal rhythm. Note absence of ectopic beats and that the *P-R* interval is now within normal limits (0.12 second).

health, gained weight, and during the period of observation had no cardiac symptoms of any kind. It is regrettable that the patient

cannot return for future study, so that it is impossible now to say whether or not the return of the cardiac mechanism to normal rhythm has proved permanent.

The preliminary interpretation of nodal extrasystoles added to nodal rhythm was based on the very short *P-R* intervals (0.10 and 0.09 second), thus fulfilling one of the two necessary requirements for the diagnosis of ectopic beats arising in Tawara's node,[3] namely, either that auricle and ventricle should contract simultaneously or that the conduction time should be distinctly shortened (normal *P-R* interval being 0.12 to 0.17 second). The other requirement—that *P*, the sign of auricular activity, should be of altered form—is only imperfectly shown in the second beat of each pair and not at all in the first. (The nervous condition of the patient rendered it impossible at any time to obtain records free from tremors.) In several cases in the literature a rhythm made up of cardiac cycles similar to the first one of each of these records has been called "nodal rhythm," in spite of the fact that Lewis and others have shown that impulses arising at an appreciable distance from the sinus node cause diphasicity or inversion of the *P* wave. A case recently observed at the Presbyterian Hospital of New York[4] demonstrates that at least some cases of apparent nodal rhythm with upright *P* are in reality due to a prolonged *P-R* interval equal in length to the interval of a cardiac cycle, so that *P* of one cycle falls on *R* of the preceding cycle. Whether true nodal rhythm can occur without obvious changes in the form of *P* is a point that is well worth studying experimentally.

In the present case, however, the gradual lengthening of the *P-R* interval under the influence of digitalis without change in the form of *P*, proves that the unusually short *P-R* interval of both cycles was due not to ectopic impulses arising in Tawara's node, but to lessened conduction time of impulses arising in the sinus node and an ectopic site in the auricle respectively. As the chief factor in the production of the *P-R* interval is the delay caused by the passage of the impulse through Tawara's bundle,[5] one must assume that the shortened interval of this case was due to positive dromotropic influences on this structure, and that these influences disappeared with repair of the pelvic disorders, as did the bathmotropic influences that caused the auricular extrasystoles. About the possible causes of such an increased conductivity, however, there is practically no evidence at hand, either clinical or experimental.

SUMMARY. A case of cardiac arrhythmia is described in which a condition of ectopic beats arising in different parts of Tawara's node was simulated by a shortened conduction time (*P-R* interval) with recurring auricular extrasystoles (pulsus bigeminus).

[3] Lewis, Thomas: Clinical Electrocardiography, p. 50.
[4] Personal communication from Dr. Strong.
[5] Hering, H. E.: Arch. f. d. ges. Phys., 1909-1910, cxxxxi, 572.

Both of these disorders of the cardiac mechanism were relieved by a pelvic operation (anterior and posterior colporrhaphy), at least to the extent that they disappeared during the period of observation.

My thanks are due to Dr. C. C. Norris for permission to study this case.

LEAD POISONING IN CHILDREN WITH ESPECIAL REFERENCE TO LEAD AS A CAUSE OF CONVULSIONS.[1]

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We are indebted to the Australian writers Gibson, Love, Turner, Breinl and Young and others for much of the recent literature regarding lead poisoning in children. They have had an unusually good opportunity to study this condition. Thus, in the Brisbane Children's Hospital, 76 cases were seen in five years and Gibson himself observed 24 cases in six years.

I shall not attempt to give a complete review of the literature on the subject, as the chief object of this communication is to draw attention to lead as a cause of convulsions in children and to report 4 cases seen in Dr. Howland's clinic at the Harriet Lane Home, the Johns Hopkins Hospital. For the more important articles, the reader is referred to those from which I have freely quoted.[2]

The symptoms of lead poisoning vary according to the susceptibility of the child and to the duration of the infection. The degree of susceptibility is variable. Breinl and Young refer to many instances in which only one child in a family was affected, although the other children were exposed to the same influences. In other instances, one child reacted with pronounced symptoms while the only evidence of infection in the others was a blue line on the gums.

Gibson[3] considers the ingestion of lead as the most likely source of infection, although he appreciates that it may occur from the inhalation of dust containing lead. He points out that children

[1] From the Harriet Lane Home, Johns Hopkins Hospital, and the Department of Pediatrics, Johns Hopkins University.

[2] Gibson, Love, Hardie, P. Bancroft, Jeffris Turner: Notes on Lead Poisoning as Observed in Children in Brisbane, Intercolonial Medical Congress of Australasia, Sydney, 1892, p. 76. Gibson, Lockhart: Ocular Neuritis Simulating Basal Meningitis Plumbism, Australasian Med. Gaz., October 20, 1897, p. 479. A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Among Queensland Children, Australasian Med. Gaz., 1904, xxiii, 149. The Importance of Lumbar Puncture in the Plumbar Ocular Neuritis of Children, Tr. Australasian Med. Congress, 1911, ii, 750. Breinl and Young: The Occurrence of Lead Poisoning Among North Queensland Children, Ann. Trop. Med. and Parasitol., 1914–15, viii, 475. Turner, Jeffris: Lead Poisoning Among Queensland Children, Australasian Med. Gaz., 1897, October 20, p. 475.

[3] A Plea for Painted Railings and Painted Walls of Rooms as the Source of Lead Poisoning Among Queensland Children, Australasian Med. Gaz., 1904, xxiii, 149.